

the grey matter, and pressure on the convolutions and deep-seated organs, which conditions were accompanied by paralysis and other signs of functional inactivity. A small patch of cicatricial tissue was found over the upper part of the right ascending parietal convolution, close to the longitudinal fissure; and we know that all his life the patient was liable to altered motility of the left side, and especially of the leg. If it could be proved that these were cause and effect, this would support recent observations by Ferrier and others on cortical localisation.

3. *The Relation between the Abdominal and Cerebral Lesions.*—Two comparatively rare diseases—namely, obstruction of the portal vein, and abscess of the brain—having occurred in the same individual, the question arises: Was each of these lesions independent of one another? or, were they in any way related? Cases are on record in which abscess of the brain seemed to have originated as a result of abdominal disease; but these are very rare, and were usually of a pyæmic nature. We have seen that the first symptoms of abdominal disease began about five months before death. Although there is no proof of it, it is possible that the abscess originated at this time. If so, could the limited peritonitis and obstructed portal system have been its cause? Of seventy-six cases of abscess of the brain, collected by Sir William Gull and Dr. Sutton, in only three could the disease be traced to an abdominal affection; and in each of these there was a collection of pus in that cavity. Suppuration in any part of the body may occasionally lead to abscess of the brain, more especially if pyæmic in character. In the case before us, there was no evidence of any suppuration having taken place, either in the abdominal cavity, or in any other part of the body; and there never had been any appearance of blood-poisoning or other constitutional disturbance, sufficient to account for the existence of a cerebral abscess. It has been suggested that this lesion was of an embolic origin, due to the abnormal condition of the portal circulation. We can, however, scarcely imagine an embolus, filtering through the liver and lungs, capable of producing mechanical obstruction; and, if the particles were sufficiently minute to effect a passage, we have no reason to suppose they should create mischief specially in the brain, unless they were poisoned, which the facts of the case disprove. In this instance, the cause of the abscess is not apparent, and therefore its age is uncertain. There was no injury, disease of the ear, or symptoms of an acute cerebral attack, which are the most common causes. Of the seventy-six cases already cited, the abscess could be traced to disease of the ear in twenty-seven; to injuries of the head in seventeen; to lung-disease in ten; to surgical affections in nine; and in eight only was no definite cause ascertained. To such so-called idiopathic abscesses, we may add this one, as the data before us fail to establish either its cause or age. We, therefore, fail to prove any relation between the abdominal and cerebral symptoms.

The facts of this case, taken as a whole, seem to suggest the following sequence of events. The patient, in early life, without apparent cause, was afflicted with chronic cerebral meningitis, which, for many years, caused no symptoms except occasional attacks of temporary hemiplegia. The changes in the tissues and circulation, thus induced, may have afterwards been the starting-point of the chronic abscess, which existed for a long time without producing any symptoms. Five months before death, the patient was seized with an acute attack of limited peritonitis, involving the portal system, and subsequently causing thrombosis and obstruction of these veins. The constitutional disturbances, induced by the second attack of this illness, probably excited the recent acute action in the brain, in the shape of cerebro-spinal meningitis, and the extension of the already existing abscess.

FUNGOSITIES OF THE FEMALE BLADDER.—Dr. Atlee publishes, in the *Boston Medical and Surgical Journal* of March 30th, a case which is specially interesting at the present time, as Sir Henry Thompson has so recently, at the Royal Medical and Chirurgical Society, drawn attention to tumours of the bladder. Dr. Atlee saw his patient in September 1880; she was a lady, aged nineteen; she was obliged to pass urine every half hour, and the urine contained a large quantity of blood. An exploration of the bladder was at once advised, and submitted to, under the influence of anæsthetics. An ordinary pair of dressing-forceps was introduced into the urethra, opened, and withdrawn; this was done several times, and the urethra thereby fully dilated. On the introduction of the finger into the bladder, no calculus or distinct tumour was to be felt; but "about the fundus were a number of fungosities, or soft growths, some of them more than a half inch in length, and about one line in thickness." Dr. Atlee scraped them away with his finger-nail, and up to the date of the paper (March 1st, 1882) the young lady has remained perfectly cured. Dr. Atlee is particular to explain that the growths were not villous, but true fungosities, having anatomically the same fundamental structure as the mucous membrane from which they sprang.

REMARKS ON ACUTE SPINAL PARALYSIS.

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We must first ask what we mean by "acute spinal paralysis". The term has become restricted to a narrower range of symptoms than it literally designates. Most acute lesions of the cord produce "acute spinal paralysis", but by the expression is commonly understood only those forms in which the paralysis is local in its distribution, and is accompanied by rapid muscular wasting. It thus includes only the disease with which we are familiar as "infantile paralysis", and the analogous affection which occurs in adults. Pathological observation has demonstrated that, in most cases, the symptoms are due to an acute inflammation of the anterior grey cornua of the cord; hence the term suggested by Kussmaul, of "anterior poliomyelitis". We may also, if we wish for a descriptive pathological name of still simpler character, call it "cornual myelitis". But it is important to bear in mind that these are pathological, while "acute spinal paralysis" is a clinical name, and they are not quite conterminous. The symptoms of acute spinal paralysis may depend on any acute process in the anterior cornua. They may occur, for instance, from hæmorrhage in this situation as well as from inflammation. We must, therefore, include under the term acute spinal paralysis, cases which are not myelitis.

The general symptoms of acute spinal paralysis are well known, since, as it affects children, the disease is familiar to all practitioners; and its characters in adults are nearly the same. There is an acute, or subacute, onset, with symptoms of a general illness. The paralysis is usually at first extensive, sometimes universal; but power gradually returns, except in a limited region, in which the muscles rapidly waste, and present the electrical reactions which characterise nerve-degeneration—loss of irritability to faradisation; preservation, and even increase, of irritability to voltaic electricity, with certain alterations in the mode of response to the latter.

Our knowledge of the nature of the process in the spinal cord is, perhaps, more meagre than in the case of any disease equally common. We know nothing of the way in which, in ordinary cases, the inflammatory lesion commences. Death during the early stage of the affection is extremely rare, and opportunities for examination have been so scanty, that there exists no observation on the state of the spinal cord during the first few days of the affection. It is possible, however, that more cases in children are fatal at the onset than is commonly believed, because the nature of the disease at this period may readily be mistaken. It is chiefly to general practitioners that we must look for the opportunity of further investigation on this important point.

It is singular that our knowledge of the causes of the disease is hardly more definite than that of its pathology. Even the causes which are recognised are not altogether beyond question. Hereditary tendency to nervous affections seems to have but little influence in its causation; most authorities doubt the influence altogether. I have myself been strongly impressed by two or three cases, in which other members of the families have suffered from other acute affections of the nervous system. This is a point on which a comparison of experience is very desirable.

The influence of age is well known, but the remarkable proclivity of children to suffer from the disease is quite unexplained. They do not suffer specially from other inflammatory affections of the nerve-tissues, although liable to certain inflammations of the membranes. The disease has been observed to come on a few days after birth. If the account given by the friends of one of my patients be correct, the disease came on, in her case, *in utero*. On the other hand, recent medical literature abounds with examples occurring in adult life. I have seen one case, in which the disease came on in a man of more than seventy years of age. After some rheumatoid pains about the neck and shoulder, he found the right hand weak; and certain muscles, especially the long extensors of the finger and thumb, and, to a less extent, the thenar and hypothenar muscles, rapidly wasted, and presented the characteristic electrical reactions. The hand also presented a well marked condition of myxœdema. I am not aware that this condition has been before observed in connection with the disease.

In young children, it is customary to refer the disease, like many others, to the irritation of teething. Is there the slightest ground for this? Here, again, we want more facts, which only the general practitioner can supply. We want to know the age and the state of dentition at the time of the onset. In a large number of cases, this information can only be given by those who have the charge of the patients.

in the early stage. Another cause to which the disease is commonly attributed is exposure to cold—sitting on cold stones and the like. That this cause is occasionally effective, can scarcely be doubted. One well marked case, in which the flexors of the hips and extensors of the knees were affected, occurred in a young lady immediately after sitting for some hours on damp grass, during the menstrual period. But it is probable that the influence of cold has been exaggerated. Many cases are ascribed to slight exposure to cold, merely because no other reason can be given. In adults, again, the rheumatoid character of the pains which attend the onset may cause the disease to be ascribed to cold, although, as is well known, in spinal disease, pain of a rheumatic character may have no rheumatic significance.

Another point in etiology which deserves discussion is, the relation of the disease to season of the year. An American physician, Dr. Wharton Sinkler, states that, on the other side of the Atlantic, three-fourths of the cases begin during one quarter of the year—between June and September. Of 149 cases he collected, 6 occurred in January, 1 in February, 11 in April, 6 in May, 18 in June, 34 in July, 43 in August, 9 in September, 6 in October, 7 in November, and 2 in December. In this country, a similar relation probably exists, since Dr. Barlow of Manchester found that, of 53 cases, 27 occurred in July and August. Hence it is clear that, whatever be the effect of cold and chills, the influence of heat in predisposing to the disease is still more potent. I have seen one case in the adult in which the disease was distinctly part of a slight attack of heat-stroke. It is very desirable that the influence of season in this country should be investigated on a still larger scale.

Among the symptoms of the disease which especially deserve attention and discussion, is the equivocal character of the symptoms which attend the onset. I venture to presume that there are few practitioners of much experience who have not, at some time, made a mistake in the early diagnosis of one of these cases. In children, the symptoms of general illness, the acute febrile disturbance, sometimes commencing with a convulsion, and reaching, as I have seen, a temperature of 103.5°, suggest an acute specific disease much more than an affection of the spinal cord; and universal paralysis has been more than once regarded as merely general prostration. In adults, the rheumatic pains are usually the obtrusive initial symptom; and the disease has repeatedly, I had almost said constantly, been mistaken for rheumatic fever. In many cases, the error in diagnosis is never discovered. When the paralysis is found out, it is supposed to have come on during the progress of a primary disease which never existed, the symptoms referred to it having really been those of the spinal affection.

This leads me to a point in etiology which I have purposely postponed, but which deserves discussion. The assumed relation to a primary disease is not always an error in diagnosis; acute spinal paralysis does sometimes come on during the course of another affection, although far less frequently than is commonly supposed. To be conclusive, the symptoms of the primary affection must be definite and unmistakable. The disease to which, in adults, acute spinal paralysis is commonly supposed to be secondary, is acute rheumatism. I believe that this opinion is usually, if not always, the result of an error in diagnosis. The only disease to which I have seen acute spinal paralysis distinctly secondary, is typhoid fever, in two cases. In the one case, many muscles of the upper and lower limbs were affected; in the other, the muscles of one forearm and hand. Each occurred about the time of puberty. The occurrence of acute spinal paralysis in typhoid fever is intelligible, since we know that, in this disease, minute foci of myelitis, which cause no symptoms, may often be found after death.

A point in diagnosis to which I would call special attention is the importance of early electrical examination. By early, I mean at the end of six or eight days from the onset. It is useless to make an examination sooner, because the indications furnished by electricity depend on the occurrence of degeneration in the motor nerves, and this degeneration occupies several days in descending from the spinal cord to the neighbourhood of the muscles. Examination with faradisation is all that is necessary at this stage. This form of electricity stimulates the muscles through the nerve-fibres contained in them; if these be degenerated, the muscles will not respond. But these motor nerves depend for their nutrition on the large nerve-cells of the anterior cornua of the cord. The fibres may be indeed regarded as the prolonged processes of the nerve-cells; as parts of the nerve-cells, and sharing every change in their nutrition; degenerating if the cells be damaged. The widely spread paralysis which occurs at the onset depends on damage too slight to cause degeneration, and it soon passes away. The muscles thus affected present no change in their reaction, and do not waste; while those which derive their nerves from the part most damaged present the reaction of degeneration, and this is followed by rapid

wasting. Thus, by finding that in some part of the paralysed region the irritability to faradisation is lost, we not only establish our diagnosis, but we can pick out and state which muscles will be ultimately affected and which will soon recover, and this before there is any other sign of difference than that afforded by the electrical examination. It may be asked, Is it safe to make an electrical examination at this early stage? Is there not danger of exciting a reflex irritation in the cord, and so increasing the damage? I believe that, if carefully done, it is perfectly safe. A very weak current will suffice, just sufficient to cause contraction in the healthy muscles; and, if necessary, the stimulation of the sensory nerves may be reduced to a minimum by employing the faradaic shock, instead of the current (which consists of a series of shocks, and is much more painful). For this, we move the hammer slowly with the finger, instead of allowing it to vibrate automatically. As an instance of the value of this test, I may mention the case of a lad, aged 16, who was seized with paralysis of both arms and legs, the onset being attended with acute fever. It was apparently due to lying with a cold wind blowing on the back, while heated after hunting. Examination showed that certain muscles of the left forearm and hand had entirely lost faradaic irritability, which was normal in the other muscles of the left arm and in both legs. Thus the diagnosis was established, and the ultimate localisation of the paralysis ascertained, although the verification of the inference was unfortunately prevented by the death of the patient, a few days later, from a cerebral complication.

The exact observation and record of the muscles affected is of great importance for scientific purposes; because we may thus obtain information, to be gained in no other way, of the anatomical arrangement of the nerve-cells representing muscles and movements in the spinal cord. Some interesting observations on this point have been published by Remak, and lately by Ferrier and Sturge; but we are still only at the threshold of the subject. As an illustration of what I mean, and as evidence that the arrangement in the grey matter of the cord is rather of movements than of muscles, I may refer to a fact which I have several times noted. Duchenne has taught us that the pectoralis major is functionally two muscles. The upper clavicular part brings the arm forward, and is associated in this action with the serratus magnus, which brings the scapula forwards. The lower sternal part of the pectoralis depresses the raised arm, and, in doing so, acts with the latissimus dorsi. In acute spinal paralysis, one part of the pectoralis is often affected without the other, and the physiological association is reproduced in the paralysis. If the upper part be paralysed, so is also the serratus, and the latissimus escapes. If it be the lower part of the pectoralis which is affected, the serratus escapes and the latissimus is paralysed.

As a rule, after recovery of the less damaged parts of the cord has taken place, a certain amount of local paralysis and wasting persists, slight or considerable. This, when improvement has ceased, remains stationary, as a rule, through life, although there may be an apparent increase in the symptoms consequent on the occurrence of deformities due to the shortening of the unparalysed opponents of the affected muscles, or on the lessened growth of bones. But I have seen two cases in which, after infantile paralysis in childhood, symptoms of slow degeneration—lateral sclerosis—came on in adult life; at seventeen in one case, at thirty-five in the other. Erb has recorded a similar case. It will be interesting to learn whether other instances have been met with by others.

Many points in the treatment of these cases deserve discussion. What is the best posture for a patient in the early stage of the disease? The extreme distension of the vessels within the spinal canal in a body which has lain on the back, familiar to all, suggests how undesirable this position is in an acute myelitis. And yet, in many cases, no other posture can be borne for long, and motion is as undesirable as gravitation. We have too often to be content with a compromise. The prone position is the most desirable; next to that, if rest can be secured in it, the lateral; the worst is the dorsal.

Are any local applications to the spine desirable in the early stage? It is only rational to treat these cases as we should treat other internal inflammations. But of the forms of local treatment—leeching, counter-irritation, warmth or cold, which is best? Much must undoubtedly depend on the patient's age and state. Counterirritation has fallen somewhat into disfavour as a means of treating the acute stage of inflammation. It is probable, however, that its *modus operandi*, and that of heat and cold, do not greatly differ; that, by each, first vascular constriction and then dilatation are obtained; and so blood-stasis is lessened, and therefore also the migration of leucocytes, which the microscope has proved to play an important part in the damage to the spinal cord from acute inflammation. But how important it is that this effect should be aided by a posture which lessens mecha-

nical congestion. I cannot but think that the damage to the cord would often be lessened, were the early treatment of these cases conducted on the principles which we apply to acute inflammation elsewhere.

Should not the internal treatment in the early stage be rather that for the morbid process than for its seat; be that for an internal inflammation rather than for a disease of the spinal cord, and therefore consist of aperients, diuretics, and agents which act on the vascular system, rather than nerve remedies? During the stage of recovery, there seems no reason to doubt that nervine tonics do some good; and on the value of electricity and skilled rubbing there is a fair agreement of opinion. When the disease was regarded as purely muscular, it was thought that electricity actually cured it in some cases. The proof that the seat of the disease is the spinal cord renders this opinion extremely improbable. Beyond doubt, the application of the voltaic or constant current to the muscles (to which their fibres will still respond, although insensitive to faradisation) improves their nutrition, lessens their wasting, and keeps them in a better condition to respond to any nerve-power over them which may ultimately be regained by the recovery of damaged cells and regeneration of degenerated fibres. Thus the ultimate state of the patient is better than it would be without electricity. It is desirable, therefore, to use such a current as shall cause slight visible contraction in the muscles. Unfortunately, children are often so much frightened by even a very weak application, that the needful strength cannot be employed without causing an amount of emotional disturbance, which may not unnaturally be regarded as doing the child more harm than the electricity does good. Here, again, we have sometimes to be content with a compromise. A weaker current than will cause contraction has probably some influence on muscular nutrition; and I think the best rule is to employ such a strength as the child will bear without much emotional disturbance, whether we can thus get muscular contraction or not. If care be taken to avoid alarming the child at the commencement, a current of some strength can often thus be employed.

Is the influence of electricity confined to the muscles? Does it aid the recovery of the spinal cord and nerves? Some believe that it does, and urge therefore that, in treating the muscles, one pole should be placed upon the spine; and the method is, on *a priori* grounds, a reasonable one. But the tendency of the disease to spontaneous improvement renders it extremely difficult to form an opinion on this point. For myself, after a careful comparative trial of the two methods of applying electricity, from the cord to the muscles, or to the muscles only, I have been unable to observe any superior advantage in the former method, which seems, even when a large spinal electrode is employed, to disturb children more than the application to the muscles only. At the same time, beyond the increased discomfort, there is no objection to the application of one pole to the spine.

How soon should electrical treatment be commenced? I cannot think it desirable to apply the voltaic current to the spine within one month of the onset; but it may certainly be applied to the muscles, with care, much sooner, at the end of ten days or a fortnight when the "reaction of degeneration" has become distinct.

ON SOME POINTS IN THE DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL DISEASE, GENERAL PARALYSIS OF THE INSANE, AND TABES DORSALIS.

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PATHOLOGICAL and clinical experience during the past ten years, in reference to nervous disease, have convinced me that, in the pathology of the general paralysis of the insane, and in the pathology of tabes dorsalis, we have an association of changes which are still open to a vast deal of investigation.

The relationship of spinal to cerebral lesions in the general paralysis of the insane has been noted for many years; but I have failed to discover any sound pathological deductions as to why the two conditions should coexist to the extent which is invariably found. We are greatly indebted to the valuable labours of Westphal, Voisin, and Mickle, for much information upon this subject. To M. Pierret we are also indebted for bringing before the profession the head-symptoms in tabes, and the important part which the trigeminal nerve takes, by its reflex influence, in the production of certain transitory pareses of the muscles of the eye, of the face, and of mastication; and also the head-pains, which are sometimes met with, but which must not be confounded with the chronic inflammatory changes of the brain

which are found in the course of the general paralysis of the insane. I have seen such cases relegated to the class of tabes, when one might as fairly have classed them with cases of gross intracranial disease, or even of general paralysis, and where, in fact, the tabes was merely a manifestation of a more or less diffuse process of degeneration, extending over the entire nervous system, although the objective signs were by comparison more marked in the posterior columns of the cord. Erb says: "Naturally, in all such cases, the importance of the spinal becomes subordinate to that of the cerebral disease." I must admit that I have had several cases under my care, where, in the initial stage (and this is extremely important from a prognostic point of view), I have been unable to give a decided opinion as to whether my patient would remain tabetic, or whether he would suddenly merge into the insane paralytic. I will give an example.

Some time ago, I was called in consultation, by my friend Mr. Willis, of Hornsey Lane, to see a patient who might fairly be said to be suffering from tabes dorsalis. He had the signs, subjective and objective, of this disease: the flying lightning-like pains, which he called rheumatic; the dysæsthesia and anæsthesia of the lower limbs; absence of knee-reflex; the Argyle-Robertson pupil; and inability to maintain the equilibrium of the body with the eyes closed; and, in addition to these, he suffered from M. Pierret's head-symptoms—namely, pains in the head involving the regions supplied by the trigeminal nerve; but there is this noteworthy fact to remember, that, during these attacks of pain, the temperature of the head was always increased. Again, his *morale* had changed, and this is not common to tabes; for, instead of being as he was formerly, an even-tempered man, he had become liable to attacks of uncontrollable excitement, and during these attacks the articulation became so markedly defective that he could scarcely be understood; but at ordinary times there was no tremor either of the tongue or facial muscles. He was a strongly built, florid-complexioned, muscular man, aged 32, without any nervous history.

I will now describe another case which has a direct bearing upon the subject. John H., aged 48, came to consult me, at the Hospital for Epilepsy and Paralysis, on December 20th, 1880. He was a florid-complexioned, strongly built, muscular man. He had suffered from shooting lightning-like pains about the body for the past ten or twelve years; they affected chiefly the anterior and the inner surfaces of the thighs and legs; they would last for five minutes, or for six, twelve, or twenty-four hours. He said these pains were not so bad as they used to be. He thought "they had given place to weakness". At times, he had burning pains of the same character in the penis, which migrated to the rectum; and these were accompanied with great desire both to urinate and to defæcate. His mental powers had failed, so that he was unable to carry on his business. When any person was talking to him, he not only forgot what he was talking about, but forgot the presence of the person to whom he was speaking. For some time, he had been unable to preserve his equilibrium, and he would roll about in walking, and his friends would accuse him of being drunk. When at the theatre, he became very emotional, and was readily excited to cry or laugh, according to the nature of the performance. Upon examining the cranial nerves, the following signs were noted. The first nerve was normal; as to the second nerve (the examination was made by Mr. Mackinlay), the discs were pale, the left one most decidedly so; the arteries small. There was no retinal change, or change elsewhere in the fundus of the eye. Right eye, vision = $\frac{2}{3}$, and C. $\frac{2}{3}$ $\frac{2}{3}$; left eye, vision $\frac{2}{3}$, and C. $\frac{2}{3}$ $\frac{2}{3}$. With the best eye, he could read Jäger 1 at a foot without glasses; the pupils were unusually small. There was no marked limitation of the field of vision in either eye; the pupils did, however, contract very slowly and sluggishly when exposed to light. The third, fourth, and sixth nerves were normal. There was marked anæsthesia and analgesia throughout the range of the fifth nerve. The lateral movements of the jaw were performed with some slowness and difficulty. He was unable to whistle or send forth a full current of air. When he talked, the mouth became filled with froth. The labials were frequently reduplicated: he pronounced "Papa's performing pony" in this way, "Pap-p-p-a perf-f-f-f-forming pony"; and, during articulation which required the exercise of labial and lingual co-ordinating power, the labial and zygomatic muscles acted without purpose. When he stood with the eyes closed, he became unsteady, and said that he felt trembling all over. The knee-reflex was scarcely appreciable. There was no ankle-clonus; no reflex of the feet. The cutaneous sensibility of the trunk was fairly normal. Muscular response was normal, both of sensation and motion, to the continuous and to the faradaic currents. There were deep-seated anæsthesia and analgesia of the lower limbs. From his own evidence, it appeared that on January 6th he completely lost his reason; and he remained in this state for a fortnight, and after this he began to improve. On July 8th, 1881, he again presented himself at the hospital,